REMARKS

After entry of this amendment, claims 41-43 and 46-47 are pending. Claim 48 is cancelled without prejudice. Claims 41-43, 46 and 47 are allowed. An amendment to claim 43 is proposed herein.

Amendment of Claim 43:

The amendment of claim 43 proposed herein adds TNFα as a protein agent that restores NFκB activity so as to treat an autoimmune disease resulting from a reduction in NFκB activity. Applicants submit that the inclusion of TNFα in this dependent claim is supported in claim 43 as filed, and in the specification at, e.g., p. 41, line 29 to page 42, line 3. Claim 43 originally listed a number of proteins, including TNFα. However, after conferring with the Examiner at the time of the Restriction Requirement, the claim was amended to recite only NFκB, NFκB p50, NFκB p52, NFκB p65 and IκB in order to facilitate examination. Applicants submit that it is widely known in the art, and directly supported in the specification (p. 41, line 29 to page 42, line 3), that TNFα induces the activation of NFκB in cells and tissues and is thus in a common pathway with the proteins currently recited in the claim. Applicants respectfully request entry of the proposed amendment and consideration of the resulting claim.

Rejection of Claim 48 under 35 U.S.C. §112, First Paragraph:

The Office Action states that claim 48 is rejected under 35 U.S.C. §112, first paragraph because the specification, while being enabling for type I diabetes, rheumatoid arthritis, Sjögren's syndrome and autoimmune hemolytic anemia, does not reasonably provide enablement for any of the other diseases recited in claim 48. Applicants respectfully disagree.

Applicants submit that the cancellation of claim 48 herein without prejudice renders this rejection moot. Applicants note, however, that in canceling the Markush claim listing specific diseases, Applicants in no way acknowledge that the scope of enablement of the claims does not extend to those diseases or others. Rather, Applicants submit that anomalous regulation of NF-κB (particularly a decrease in NF-κB activity or a failure to increase NF-κB activity in response to a stimulus) has been positively associated with the pathology of many autoimmune diseases

having different disease etiology, thereby supporting Applicants' conclusion that anomalous NF-κB regulation or responsiveness is a central factor in autoimmune disease in general. In the interest of obtaining protection for the claims deemed allowable in the present Office Action, Applicants have herein canceled claim 48, and intend to continue the pursuit of protection for claim 48 in a continuing application.

In view of the above, Applicants submit that all issues raised in the Final Office Action are addressed herein. Applicants respectfully request entry of the amendments proposed herein and reconsideration of the claims.

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Respectfully submitted,

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Version of Amendments Marked to Show Changes:

43. (Twice amended) The method according to claim 42, wherein said protein is selected from the group consisting of: NFκB, NFκB p50, NFκB p52, NFκB p65 [and] , IκB and TNFα.